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An Account of the Atlantic City Meeeting

FELLOW MEMBERS OF THE GASTROSCOPIC SOCIETY:

Though we had 63 members and guests present at the scientific session at The Claridge Hotel in Atlantic City on April thirtieth, it has seemed to me that those members who were unable to attend might like to know what went on there. Those of you who saw my harrassment in getting lanterns into operation and in darkening, and yet ventilating, the Hotel's Solarium, will understand why I may have missed some important points. Should that be the case or if I have misunderstood the speakers, I'm certain that clarifying letters to The Bulleton would be published.

We got off to a late start with our scientific session because the Hotel hadn't attended to details in the meeting room very well. But with Dr. Patterson in the chair things moved along smoothly and no time was wasted after we were under way.

The first paper was that of Dr. Wirts and of Dr. Cimoch, Philadelphians, on "The Use of Intravenously Administered Demerol for Gastroscopy". Dr. Wirts said that they have been using from 50 to 100 mgm. of Demerol intravenously immediately prior to gastroscopy. I believe that their average dose was from 75 to 100 mgm. This had been administered with the patient lying on the operating table because it has acted so very rapidly. Frequently it has put the patient into a semi-sleep in which he has been fully cooperative. With its use no gargle or cocaine or of Pontocaine has been necessary. In 15 to 45 minutes the patient has usually recovered from the effects of the sedation and he is kept in the hospital for an hour or two before he is sent home. The unpleasant side effects had been minimal ones and I noted that the principal distressing symptoms were nausea, dizziness, and euphoria.

Dr. Boros asked Dr. Wirts whether or not the effect of Demerol given intramuscularly approached that of the intravenous administration of the drug. Dr. Wirts replied that it should be given intravenously since its effect was more profound that way.

Dr. Donovan Browne inquired concerning the length of the sedation and my notes say that Dr. Wirts replied to the effect that about one-third of the subjects got very sleepy and that the drowsiness persisted for about forty minutes on the average.

Dr. Donald Marion asked whether or not the an-

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Diagnosis of Gastric Bleeding

Case Report Illustrating the Value of the Gastroscope

HENRY LAURENS, JR., M.D.
Ada, Oklahoma

Mrs. G. C., a sixty-year old housewife was admitted to the hospital on June 27, 1952, several hours after she had suddenly become sick and vomited a considerable amount of bright red blood. Shortly after the hematemesis, she passed several large, tarry stools. She complained of being very weak, faint and thirsty.

Examination on admission revealed an acutely ill, anxious, very obese female who weighed 247 pounds. The blood pressure was 140 systolic and 100 diastolic. The pulse was slightly weak with a rate of 90 per minute. Examination of the eyes, ears, nose, and throat revealed no abnormalities. Examination of the chest was negative. The heart was normal except for somewhat distant tones and a rate of 90. The lungs were clear to percussion and auscultation. Examination of the abdomen revealed no abnormalities. There were no masses and there was no tenderness. There was no evidence of abnormal collateral circulation. There were no telangiectases. Examination of the rectum was normal except for the presence of tarry stool.

On admission her red blood count was 2,950,000, hemoglobin 64%, white blood count 9,300 with a differential of 94 segs and 6 lymphocytes. The urine was perfectly normal. Following admission she was given sedatives, intravenous fluids, and two blood transfusions.

Several hours after admission, the patient was able to give a history of having had four previous hemorrhages which were quite similar to the present illness. The first of these attacks had occured in 1941, at which time x-rays, which were unfortunately lost, were reported to have been negative. The last previous attack had occurred in November, 1949. On each previous occasion she had vomited a considerable amount of bright red blood which was followed by tarry stools. However, on each of these occasions she was treated with bedrest, diet, and an iron preparation, and since she always responded quite well to this treatment, and the previous x-rays demonstrated no abnormalities, she and her family were reluctant to accept surgery. On each occasion she stopped bleeding after the initial episode and had no further difficulty until the next attack of

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An Account of the Atlantic City Meeting

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aesthesia produced such relaxation that the patient would not hold air pumped into his stomach. Dr. Wirts said that there was not excessive belching with intraveously administered Demerol.

Dr. Sexton asked if it was necessary to give the patient barbiturates as a preliminary to Demerol in the vein. Oversedation resulted from such a procedure, according to Dr. Wirts.

Dr. Wirts said in reply to a question about the rate at which Demerol is injected, "2 cc. of Demerol Solution are put in a 10 cc. syringe and, after the vein is entered, 8 cc. of blood are aspirated into the syringe. The mixture of Demerol and blood is then injected slowly."

Dr. Patterson asked for a show of hands by those men present who knew from reliable sources of deaths following the use of Pontocaine. I was surprised to see 15 hands raised, for I've had no trouble with Pontocaine when a barbiturate was given intramuscularly prior to its use.

Then followed a discussion called Problems in Oesophagoscopy", Dr. Moses Paulson opened that. He said that he considered the oesophagus to be the province of the gastroenterologists, a province which the otolaryngologists had developed because of the gastroenterologists' neglect of the field. He urged us to learn oesophagoscopy.

I told the meeting that I had recently had a difficult oesophageal problem in a twenty-two-year-old boy who was a victim of cerebral spasticity, a condition which had begun at birth. He had athetoid movements constantly, extreme scoliosis of the spine, and a short oesophagus with a supradiaphragmatic stomach of fair size. He lived in Argentina and he had come to The States because of an oesophageal stricture which must have developed on the basis of peptic oesophagitis. Physicians in Buenos Aires and in New York had declined to make oesophagoscopic examinations or to attempt dilatation of the stricture because of the patient's muscular rigidity and scoliosis. With phenobarbital sodium and morphine and repeated assurances, I was able to make a satisfactory oesophagoscopic examination with the Jackson 'scope. The stricture was clearly seen and it was fiery red. There were patches of white exudate on its edges. It was clearly a benign affair. Dilatations with a Broyles dilator and subsequently with beads passed over a previously swallowed thread were not so simple. It was necessary to anaesthetize the patient with sodium pentothal and curare and we introduced intratracheal tube. Then the dilatations were carried out on the fluoroscopic table to be certain that the dilators passed below he patient's diaphram. The stricture was a very tight one and it was stretched to a No. 45 French size in two sessions. The subject was relieved of his dysphagia, though he has continued to have heartburn.

Dr. Boros said that he had seen sword swallowers perform on a recent trip to Egypt.

Finally, Dr. Patterson gave a word of caution about the use of rigid open-tube oesophagoscopes.

The next subject for discussion was "The Clinical Significance of Superficial and Hypertrophic Gastritis." Dr. Eddy Palmer pointed out that biopsies taken from gastric mucosa which showed the typical gastroscopic picture of hypertrophic gastritis were often microscopically normal. He suggested that the pebbled appearance of the stomach might be the result of contractions in the muscularis mucosae and that the clinical symptoms of such gastritis *could* be the result of a muscular difficulty in the stomach. Dr. Palmer regarded chronic superficial gastritis as a precursor of gastric atrophy and he pointed out that such gastritis might be a source of gastric hemorrhage. The conclusion was that we don't know enough about gastritis.

Dr. Frank McGlone stated that the French physicians have *thought* that they knew a good deal about gastritis. I got the idea that he felt that the French actually knew relatively little about the subject.

Dr. Sexton, who has known Dr. Moutier personally, stated that the great Frenchman hadn't been able to see under the microscope the gastritis which he had observed with the gastroscope. He said that Dr. Moutier had the concept that the differences in the appearance of the tissue had something to do with its fluid content when it was in situ and after excision. Dr. Sexton reminded us that the French physicians feel that unhealthy changes in the gastric mucosa always precede the development of malignancies there.

Dr. Donovan Browne pointed out that the gastroscopic appearance of hypertrophic gastritis may change in a striking way over a few days time.

Dr. Shallenberger agreed with Dr. Palmer in his statement that biopsies from hypertrophic gastritis were frequently histologically normal. He stated that in his opinion Dr. Moutier's idea of fluid changes in biopsied tissue was incorrect. He reminded us that the time from the taking of a biopsy until its fixation was remarkably brief.

Dr. Hardt pointed out that hypertrophic gastritis may stimulate malignancy as seen through the gastroscope and he confirmed Dr. Browne's remarks when he said that two weeks of a soft diet would change the gastroscopic picture of hypertrophy and inflamation to normal.

Dr. Patterson stated that a shaggy gastric mucosa sometimes occurred in the fundus of the stomach and that it was called a "cow's tit formation". He said that it occurred in heavy coffee drinkers and persons who ingested irritants. He thought that bleeding might come from such a process. He (or perhaps it was someone else) estimated that hypertrophic gastritis was diagnosed in less than 10% of patients examined with the gastroscope.

Dr. Flood said that the gastroscopic and clinical pictures in gastritis frequently could not be correlated and he wasn't too certain that the gross appearance of the gastric mucosa was too significant.

Dr. Rumball remarked that we might have to reclassify our ideas of gastritis.

Dr. Palmer closed by reminding us that the military services and the book of diagnosis of The A. M. A. recognize chronic hypertrophic gastritis. It is an "official diagnosis."

Then Dr. J. Edward Berk opened the discussion on "The Lesions of Hiatal Herniae". His text was, "Beware of Greeks bringing gifts!" This meant that the very presence of a hiatal hernia did not always mean that it was the source of bleeding when one was confronted with a problem of upper gastro-intestinal haemorrhage. He reminded us that, when bleeding occurs in a patient with a hiatal hernia, peptic ulceration outside the hernia may be demonstrated in about 10% of the cases.

Dr.Kushlan said that he had had trouble seeing the interior of hiatal herniae gastroscopically. To this Dr. Berk replied that he thought that the flexible gastroscope showed most sizeable hiatal herniae quite well. Someone stated that the oesophagoscope gave the best view of a thoracic stomach but I got the opinion that that idea wasn't generally shared.

Dr. Brick pointed out that the association of peptic ulceration with hiatal hernia (ulcers outside the herniated stomach) was greater than 10%. He pointed out that many persons had hiatal herniae without symptoms.

Dr. Berk stated that, in his opinion, when ulcer was present in a hiatal hernia, it was usually at the hiatus. He believed that its surgical treatment with phrenic crush or with gastric resection was rather successful.

Dr. Berk's remark was in answer to a question of Dr. Rothmiller who wondered how many patients had the ulcer in the hernial sac.

Dr. Flood was of the opinion that most of the ulcers of hiatal herniae are at the cardio-oesophageal junction. He felt that the bleeding from hiatal hernia was generally mild.

Dr. Wirts felt that the pouch of hiatal herniae can be seen quite well with the flexible gastroscope and he has visualized ulcers in the pouches. He seemed to be inclined to have the thoracic surgeons repair the herniae for the treatment of such ulceration.

Dr. Emmanuel Deutsch of Boston opened the subject of "Gastroscopic Biopsies". He had observed that the presence or absence of changes in the tone of the gastric musculature had been useful in differentiating benign from malignant ulceration. Additional aids in this differential diagnosis were gastric biopsies taken through the Benedict Operating Gastroscope and the study of gastric cytology. The material for this latter study was obtained by gastric aspiration following the scraping of the stomach wall with a polyethylene tube.

Dr. Oren showed an ingenious brush for obtaining from the stomach material for cytological study. This had been devised recently by Dr. Ayre of Miami and it had been used most successfully by Dr. Oren. The apparatus was essentially a plastic tube of the approximate calibre of a Levine tube through which was run a flexible rod. This tube could be passed into the stomach and positioned, if desired, with the fluoroscope. By pushing downward on the flexible shaft, two rather coarse brushes could be projected from the tube's end and these could be rotated in the stomach as they were withdrawn. Thurs the gastric wall might be "swept". Before the tube with withdrawn, the brushes could be pulled back into its lumen so that material gathered in the stomach would not be scraped off in the gullet. After the tube had been recovered completely by the examiner, material from the brushes was transferred to slides.

At the conclusion of Dr. Oren's remarks, Dr. Patterson brought the scientific session to a close. A brief business meeting followed.

The Secretary-Treasurer reported that we had 166 members on April twenty-ninth, excluding the three men who had resigned from the Society in the past year. At the meeting of the Governing Board, which immediately preceded the scientific session, five candidates were admitted to membership and two others will probably be added to that number when technicalities associated with their applications have been complied with. So we are a growing organization.

The financial condition of the Society was reported and the membership seemed to consider our economic status to be a healthy one. The books and accounts of the Secretary-Treasurer were approved by an Auditing Committee of Dr. D. F. Marion, Dr. Frank B. McGlone, and Dr. Leo Hardt.

A Nominating Committee headed by Dr. Eddy Palmer presented the names of Dr. H. Marvin Pollard for the Society's president, Dr. Marie Ortmayer for vice-president, and Dr. John Tilden Howard for secretary-treasurer. There were no nominations from the floor and the candidates recommended by the Nominating Committee were unanimously elected.

Dr. Kirsner moved that the interest on the money in the savings account of the Society be used to establish a Gastroscopic Award. This would be given annually by the Society (on recommendation of a Committee) to the person who had contributed most to gastroscopy in the preceding year. Dr. Kirsner stated that the Committee should have the privilege of withholding the award if in any year no noteworthy contribution to the specialty was made.

In discussing the motion, Dr. Kirsner pointed out that his proposal kept control of the award in the Society which had contributed the money that made the award possible.

Dr. Kisner's motion was seconded and it was passed unanimously.

The business meeting was then adjourned.

Our cocktail party followed the scientific session immediately. It was held at the entrance to The Claridge's East Room, where we sat down to dine a half hour or so later. The cramped quarters for the party didn't interfere with its success; we literally "got together". In our small group we are generally known to each other and everyone seemed to have a good time.

Fifty-four members, their wives, and guests attended the banquet, which was skillfully handled by the Hotel. The food was really good and the service was excellent.

After dinner Dr. Patterson introduced Dr. and Mrs. Eusterman, who had drifted in toward the end of our meal. And he asked the new officers of the Society to take bows.

Dr. Pollard showed Kodachromes which he had taken when he was in Europe last summer and he spoke particularly about the British medical situation. I got the impression that the teachers and full-time workers in hospitals had spoken very favorably of State Medicine to Dr. Pollard and that doctors who were not in the governmental organization of physicians were just as opposed to the new regime. Dr. Pollard said that the British physicians in the governmental organization hospitalized all their patients with peptic ulceration and that the patients were kept in bed for three or four weeks. During the first week of their treatment they were given intra-gastric drips routinely. When Dr. Pollard had enquired concerning the need for such strict therapy and when he had pointed out the fact that most ulcer patients were treated on an ambulatory programme in this country, the British State medicos replied to the effect that their system enabled them to give rich and poor what they considered to be the very best therapy, something which was not available to ulcer patients in the lower economic brackets in The States.

Though it was late when our official gathering broke up, there was much talk in the Hotel's Mayfair Lounge and in the members' rooms over drinks 'til early morning. 'Twas a well attended and an enthusiastic meeting, scientifically and socially.

JOHN TILDEN HOWARD

Diagnosis of Gastric Bleeding

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bleeding. However, her stools always remained tarry for several days following the onset of bleeding.

On November 2, 1949, the date of her last previous episode, the patient had been admitted to the hospital. On that occasion an x-ray of the upper gastro-intestinal tract was attempted, but she was too weak to stand and the x-ray apparently was unsuccessful. A barium enema done at that time was negative. Following the initial bleeding episode she began to pass some bright red blood from her rectum, and a large left inguinal hernia,

which she had had for thirty years, became incarcerated. Therefore, at that time it was thought that some of her bleeding originated low down in the bowel, and since all of her bleeding stopped after the hernia was repaired, she was discharged from the hospital with no further studies being made. Following the episode in 1949 she was perfectly well until her present admission to the hospital. She had no indigestion, could eat anything she wanted, and had no nausea or vomiting. She did state that for several days prior to her present bleeding episode, she had been doing some rather heavy lifting around her house.

X-ray examinations of the upper gastro-intestinal tract on June 29, 1952, was reported perfectly normal except for a large diverticulum on the medial side of the second portion of the duodenum. There was no evidence of any diverticulitis. (Fig. 1.)

FIG. 1—Arrow points to area of decreased density in antrum.

Subsequent laboratory examinations revealed a thymol turbidity of 4.8 units, bromsulfalein retention of 0.5%, direct bilirubin 0.10 mg., total bilirubin 0.25 mg., NPN 66.5 mg., bleeding time two minutes, clotting time two and one-half minutes, platelet count 140,280, prothrombin 51%, and a negative serology. Initial gastric analysis revealed no free hydrochlōric acid, but following histamine there were 32° of free acid and 60° of total acid.

The patient continued to pass tarry stools and it was necessary to give her at least 500 cc. of blood daily in order to maintain her blood count. However, on July 3, 1952, her red blood count did seem to become stablized at 3,930,000 with a hemoglobin of 12 gm. or 78%.

On July 3, she was gastroscoped without any difficulty, and this examination revealed immediately the cause of the bleeding. An excellent view of the angulus and of the antrum was obtained, and situated on the lesser curvature of the antrum, just proximal to the pyloric sphincter at approximately the 12 o'clock position, there was a fairly large, angry, red polyp. This polyp appeared to be approximately ½ cm. in diameter and was definitely pedunculated. As the pyloric sphincter opened and closed, the polyp at times disappeared through the pylorus, and definitely seemed to be squeezed upon by the pyloric sphincter. However, during the examination, no fresh bleeding was observed. The gastric mucosa surrounding the polyp appeared to be perfectly normal, but higher up in the stomach, along the lesser curvature of the body, there were several areas of definite cobblestone formation suggesting early hypertrophic changes.

She was given several more blood transfusions, and on July 5, 1952, gastrotomy was performed. At surgery, a pedunculated polyp which measured 1x1½ cm. in diameter was found on the lesser curvature of the antrum, just proximal to the pyloric sphincter. The polyp presented definite evidence of recent hemorrhage. In addition to this, a very small 3 mm. sessile polyp was situated about 6 cm. proximal to the polyp. Both polyps were removed together with a surrounding rim of normal gastric mucosa and the stomach was closed. Pathologic examination revealed benign gastric polyps with no evidence of malignancy. (Fig. 2.)

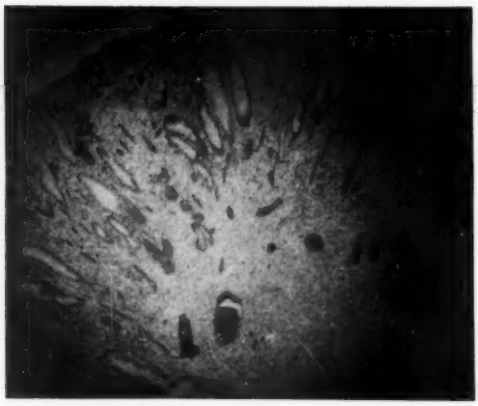

FIG. 2—Pathological examination revealed benign gastric polyps. No evidence of malignancy.

Post-operatively the patient did very well. On July 11, she did pass a large tarry stool, but this was believed to be due to residual blood within the intestinal tract. She did become nauseated and vomited on several occasions after she began to eat solid food, but when she was advised to turn on her right side and abdomen following meals, so that her diverticulum might empty in case it was retaining food, she became asymptomatic.

Following up course reveals that she has had no further difficulty. She was seen on August 4, 1952, at which time her weight was 237 pounds, physical examination was entirely negative, hematocrit was 44, RBC 4,350,000 with hemoglobin of 89% or 12.7 gms. The stool was negative for occult blood. She was next seen on September 9, 1952, at which time she had no symptoms and the stool was negative for occult blood. She was next seen on October 7, 1952, at which time she had no symptoms, her physical examination was normal, hemoglobin 97%, RBC 4,300,000, and stool negative for occult blood.

The patient was last seen on January 13, 1953, at which time her weight was 228 pounds, she had no complaints, physical examination was perfectly normal,

stool was negative for occult blood, and very thorough fluoroscopic and roentgenologic studies of the upper gastro-intestinal tract revealed no abnormality except for the previously described duodenal diverticulum. (Fig. 3.) In retrospect, review of the pre-operative x-rays, especially when compared with the post-operative films, suggests that there was a small persistent filling defect in the pre-pyloric antrum.

FIG. 3—No abnormality except for previously described diverticulum.

COMMENT: This patient is a very good example of the importance of gastroscopy in the diagnosis of upper gastro-intestinal hemorrhage. It was thought at first, when the duodenal diverticulum was noted, that this might be the source of her bleeding, though the rarity of the importance of this lesion as the source of upper GI bleeding was well appreciated. There is no doubt that the polyp could have been diagnosed in 1949 if gastroscopy had been performed even though the patient was too wyeak to undergo x-ray of the upper gastro-intestinal tract. It is well recognized that with the proper psychological approach, a gastroscopic examination under such emergency conditions can often be done without any risk to the patient and without any aggravation of bleeding.

It is interesting to note that this patient's polyps existed in gastric mucosa which was not atrophic. Though the gastric mucosa was slightly hypertrophic in some places, and there was an adequate amount of free hydrochloric acid present, there is no doubt that the large polyp was not an inflammatory one. Gastroscopy was especially valuable in this case of a very obese women who was very difficult to fluoroscope due to her obesity, and could not be subjected to prolonged x-ray examination due to her acute illness.

SUMMARY: A case is reported in which the diagnosis of massive upper gastro-intestinal bleeding, which had occurred on five occasions over a period of twelve years, was made only when gastroscopy revealed a benign gastric polyp showing evidence of recent hemorrhage.

A Congress on Chronic Gastritis

By ROGER CATTAN

Paris

The 37th Annual Meeting of the Radiological Society held in Chicago in December 1951 had on its program a study of chronic gastritis. This is a question dear to French gastroenterologists and we were very glad to have had this opportunity to become acquainted with what they are thinking about on the other side of the Atlantic. Let us say immediately that this reading took us back many years to our first steps in the radiology of the digestive tract.

The first paper was entitled "A revue of the literature". This revue includes only two French names—illustrious but old—Broussais and de Hayem. A congress as important as that of Brussels in 1935, the first held by The European Society of Gastroenterology which was devoted to gastritis, is not even mentioned. What a rich material the American writers could have found in the remarkable papers of Moutier, of Lion, of Hurst, or Konjetzny, of Zweig, of Paschoud, etc.! What precise answers to questions about diagnosis, pathologic anatomy, and treatment of gastritis!

The second paper was by H. H. Berg. He discusses antral gastritis and is certainly the most solid of all. However, he does not tell us anything new. A long time ago agreement was reached in France on the classification, the pathologic anatomy, the pathogenesis and the clinical symptoms and radiologic signs of these fibrous inflammations of the antrum. Is it possible that Berg allowed to escape such an important reference as the article by Guy Albot and Francois Magnier who in 1948 brought to this question a decisive contribution supported by unchallengeable arguments based on radiologic and anatomic evidence? (Arch. des Maladies de l'App. digest., Sept.-Oct. 1948, 37, 560-587.) Could he be equally unaware of the important European bibliography which followed it?

The difficulty of the radiologic differential diagnosis between antral gastritis and cancer seem to have been the principal preoccupation of the authors. Several of them made this particular point the center of their discussion. Berg, for example, enumerates the principal differential signs: abrupt interruption of the folds, oblitration of peristalsis, constancy of the deformity in cancer, etc. The importance of careful radiologic study of each individual case will not be denied in France. It was a long time ago that Gutmann taught us to investigate "as with a magnifying glass" the slightest deformation of the lesser curvature and that we have known that the antral deformities "reactionelles", "organofonctionelles" are extremely frequent.

In an attempt to clear up this problem, the Parisian school of gastroenterology, with Porcher, Gally, Albot, invented pharmacoradiology of the stomach in which the use—depending upon the case—of morphine subcutaneously or more rarely intravenously, of ipecac, of insuline, increases the tonus and the contractions of the stomach at the same time that the duodenum dilates. In addition one tries to modify the gastric contours with atropine.

Under the influence of these new methods of investigation (of which the morphine test of Porcher is justly the best known) one sees the contours in antral gastritis completely changed, while those of cancer remain unchanged. Doubtful cases will be considerably reduced in number. Most often the diagnosis is easy.

Well, unlikely as it may seen to us, pharmacoradiology of the stomach seems to be completely unknown to the writers of this Radiological Society meeting.

Neither Berg, nor Stampfli, who made a study of the mucosa in gastritis, nor MacGlone, nor Kennedy referred to it. Only Ross Golden, incidentally in the discussion of these papers, said 'to increase gastric peristalsis, French workers used intravenous injections of morphine."

It is not less astonishing to note that mention is not made to other methods of radiologic examination currently used in Europe; nowhere, for example are compression films mentioned.

Frank MacGlone, in regard to hypertrophic gastritis of the cardiac portion of the stomach, says, in so many words, that the diagnosis is most often impossible. For several years we in France have tried, nevertheless, to make it. Various methods of gas distention are used, and recently Porcher has suggested that they be combined with pneumoperitoneum. He thus obtains magnificent direct shadows of the stomach wall and, in the case of gastritis, the disappearance of "false" defects.

I do not assume that this Radiological meeting has undertaken the task of examining all aspects of gastritis. In that case, much could be said about the total omission of what Moutier called the "gastrides" and the "apogastritis", that is to say, in contrast to the infectious or toxic gastritis, the gastropathies of allergic, dystrophic or deficiency state origin. It would have been interesting to consecrate a few lines to the radiologic aspects of the stomach in pernicious anaemia.

In consideration only of that which we read, we cannot allow to pass without a protest the report of Ingersol on the probable pathogenesis of chronic gastritis. This author assumes that herniation of the prepyloric mucosa through the pyloric canal is invariably present at the beginning of all gastritis and is the real cause of it. Certainly we know that the authors on the other side of the Atlantic attribute more importance than we do to this anomaly but nobody has ever gone so far in the interpretation of the difficulties it can cause. For him, in fact, a succession of incontrovertible facts exist: the first is this famous prolapse or, better invagination, of the prepyloric mucosal folds into the duodenum. Then antral gastritis develops which becomes generalized secondarily; finally fibrous antral gastritis terminates this evolution.

This conception goes counter to everything we know about gastritis. First, this fact, recognized by all the endoscopists, that gastritis of the body of the fundus and of the region of the angle is much more frequent than that of the antrum. Secondly, that antral gastritis (or better designated, as suggested by Gutmann, by the name "antral reaction" to avoid the assumption from a radiologic abnormality that a histologic lesion is present) is most often secondary to an ulcer of the lesser curvature. Finally, transpyloric invaginations are far from being constant. The ideas of the American writer appear to rest on rather flimsy arguments.

How willingly we agree with Vaughan's ironic criticism of Kennedy's paper. Does not the latter believe that gastrectomy should routinely be done whenever the [illegible]um is under suspicion? He thinks, in fact, that the differential diagnosis of cancer and gastritis is impossible, clinically as well as radiologically, and even to the surgeon with the stomach in his hand. He advises resection of the lesion, and if immediate frozen sections show cancer to do, secondarily, further resection. How far we are from the minute study of 16 to 24 exposures which we are accustomed to make, from our pharmacoradiologic investigation, and above all, from our "therapeutic tests"! Under the decisive influence of R. A. Gutmann, immediate gastrectomy is hardly ever done in France except for lesions which are definitely cancer. If the slightest doubt exists about the benign character of the lesion the test of therapy is begun: Three weeks of carefully controlled treatment in which protein therapy is the most important part. At the end of this period complete examination of the stomach is done again. If the lesion has progressed or has remained unchanged, intervention must be advised. In the contrary case (presumably evidence of improvement) intervention can be avoided. Light-heartedly to do a gastrectomy for a lesion which is possibly inflammatory and edematous appears to us inadvisable. The risk of the intervention itself and of its sequellae is apparently not understood, which appears to us greater in gastrectomy for gastritis than for a limited ulcerous lesion. How much more sensible appears to us the report of Paschoud on the same subject which was given at the first European Gastroenterological Congress in 1935.

French physicians have often been reproached for not paying attention to foreign publications and for not keeping a bibliography. It appears that this reproach is no longer justified. On the contrary, our American colleagues would have everything to gain by trying to decipher the work written in a language which was formerly that of learned men and literateurs: the French.

The Lesions of Hiatus Hernia: The Co-Existence of Hiatus Hernia And Gastroduodenal Ulcer*

By J. EDWARD BERK, M. D.

The broadness of the topic to be discussed and the limitations of time prompt me to confine my opening remarks to one phase of the subject. My message is simple in the extreme and my text is taken from Vergil's Aenead in which is recorded Laocoon's memorable warning to his compatriots, " . . . timeo Danaos, et dona ferentes" "I fear the Greeks, even bearing gifts."

From August 1948 to March 1950, a 56-year-old, obese, hypertensive, white female was followed in the Gastro-Intestinal Clinic of Temple University Hospital. For ten years prior to the time she was initially seen she had been having substernal burning. During the year immediately preceding her first visit to the clinic she had occasionally vomited material containing flecks of blood and had also noted that her stools at times were black. Studies of the gastro-intestinal tract disclosed only a large hiatus hernia. During August 1948 a massive gastro-intestinal hemorrhage occurred that was attributed to the hernia. After consultation with the surgical department, it was decided to persist in the use of conservative medical measures because of the patient's obesity and hypertension.

Throughout the subsequent period that the patient was observed in the clinic, she continued to have recurring epigastric distress. Finally, in March 1950, she developed severe epigastric pain. This was shortly followed by hematemesis and melena which persisted despite treatment and resulted in her death on March 12, 1950.

Autopsy showed that a considerable portion of the upper stomach had herniated into the left pleural cavity. At a point about 3 cm. below the cardiac orifice, the

* Abstract of remarks made at the annual meeting of the American Gastroscopic Society, Atlantic City, N. J., April 30, 1953.

lesser curvature of the stomach was found to be adherent to the left lobe of the liver. When the stomach was opened, a chronic ulcer was noted at the point of adherence. The ulcer measured 3.8 cm. in diameter and was found to have perforated the entire gastric wall to become adherent to the liver. In its base was a large blood vessel, the superficial aspect of which was eroded forming an opening approximately 3 mm. in diameter.

It was quite obvious from these findings that the patient had bled not from her hiatus hernia per se but from the associated gastric ulcer. In restrospect, too, it appeared likely that many of her symptoms were also to be attributed to the gastric ulcer rather than to the hernia. This experience caused us to question how frequently massive gastro-intestinal hemorrhage occurs in patients with hiatus hernia. It also stimulated us to inquire how often chronic gastric or duodenal ulcer is found in association with hiatus hernia.

Accordingly, Dr. Herbert Hyman, at that time Fellow in Gastroenterology, undertook to survey our experience at Temple University Hospital and to review the more recent pertinent American literature on the subject. It immediately became apparent that it would be difficult, if not impossible, to obtain accurate answers to our questions from available reports in the literature. The principal reasons for this were pronounced variation in the criteria for gastro-intestinal hemorrhage and failure of some authors to describe specifically the anatomical location of the peptic ulcers noted by them to co-exist with the hernia. As a consequence, our data are still in process of modification and must be considered subject to future revision.

The mean incidence of significant gastro-intestinal bleeding in patients with hiatus hernia as presently established by Dr. Hyman from 26 reports dealing with 3061 cases (including our own material at Temple University Hospital), was 25.2%. Included in this computation were patients described as having hematemesis, melena, or simply "hemorrhage". Not included were patients with anemia alone. This required omission of a sizeable number of cases because in several reports patients with anemia, hematemesis, melena, or positive stools for occult blood were inseparably grouped in the single category of gastro-intestinal bleeding. Also excluded from the group with gastro-intestinal hemorrhage were patients whose sole evidence of bleeding consisted of positive reactions for occult blood in stools. It was impossible, however, to distinguish all of the latter from those with true melena since many times patients with either of these phenomena were combined and not separately classified.

The mean incidence of gastroduodenal ulcer, based on 18 reports dealing with 1192 cases (including our own series at Temple University Hospital), was found by Dr. Hyman to be 10.7%. The latter incidence represents a combination of the recorded incidence of specified duodenal and gastric ulcers and of unspecified "peptic" ulcers.

The high mean incidence of significant gastro-intestinal hemorrhage (approximately 25%) is impressive and needs no further comment. The mean incidence of gastroduodenal ulcer would appear to be no greater than that for the maximum estimated incidence of peptic ulcer in the population at large. However, when it is considered that hiatus hernia is frequently seen in obese females among whom duodenal and gastric ulcers are less frequent than in other segments of the population of comparable age, the mean incidence of almost 11% of gastroduodenal ulcer in patients with hiatus hernia acquires added significance.

The lessons to be learned from these data, it seems to me, are that significant gastro-intestinal bleeding occurs surprisingly often in patients with hiatus hernia, and that hemorrhage, particularly when appreciable, may originate from a co-existent gastric or duodenal ulcer and be independent of the hernia itself. When one encounters gastro-intestinal bleeding in a patient known to have a hiatus hernia, one naturally thinks of mechanical and vascular alterations common to the hernia as the probable responsible factors. However, when it is recalled how frenquently chronic gastric or duodenal ulcer may co-exist with hiatus hernia, it becomes important to exclude the existence of such lesions before concluding that the hemorrhage is due to the hernia alone. Indeed, it would seem wise in these circumstances to adopt toward the hernia an attitude of distrust similar to that of Laocoon who warned of his enemy, . . . "timeo Danaos, et dona ferentes" "I fear the Greeks, even bearing gifts".

SYNOPSIS

The Significance of Hypertrophic and of Superficial Gastritis

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The criteria for gastroscopic diagnosis of chronic hypertrophic gastritis have been rather well standardized, and the probable significance of the condition as a clinical entity has received tentative acceptance in many clinics. The vehemence of speculation noted in the literature of only a decade ago has suddenly become quiet, not apparently as a result of declining interest but as a consequence of accumulating objective information. The advent of simple gastric mucosal biopsy technics appears destined to create once again turmoil among those who have a special interest in the gastritides.

In his monumental studies Dr. Schindler has offered as a histopathologic explanation for hypertrophic gastritis a set of changes presented as both outspoken and constant, as judged by material removed at operation. His correlation of gastroscopic and histologic classifications has been accepted at face value by many gastroscopists. Occurrence of the three histologic varieties as described by Dr. Schindler has been confirmed from time to time. But, although glandular and foveolar hyperplasia are well known to the pathologist, they are encountered uncommonly—too much so to correlate with the frequency of hypertrophic gastritis claimed by the gastroscopists. Another basis for confusion in the mind of the clinician is the irreconcilibility of the seemingly static histologic nature of the proliferative and glandular types, with the quick and marked gastroscopic changes often reported in the literature. There can be litte question that either the gastroscopic interpretations are erroneous in such instances or that there is poor correlation between accepted gastroscopic interpretation of the gastritides and current histopathologic explanations for the gross change..

PRESENT OBSERVATIONS

In the course of the last 2500 gastroscopic examinations performed at this hospital, the diagnosis of chronic hypertrophic gastritis was made in 190 instances. Mucosal biopsies were taken by vacuum-tube technic in several cases. Fifty-three speciments from 43 patients had been removed from areas which upon immediate gastroscopic examination were found to show chronic hypertrophic gastritis. Among the 53 speciments, disease could be found histopathologically in only 11. In none was the disease more than relative and notably mild. No glandular hyperplasia, foveolar hyperplasia, or excessive infiltrative disease was found.

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COMMENT

In normal people during the fasting state the degree of round-cell infiltration into the mucosa's interstitium appears to be rather minor. To some experienced pathologists, any excess in the amount of infiltration constitutes the only abnormality required for the diagnosis of chronic gastritis. By this concept, all chronic gastritic is taken to be one pathologic process, sometimes with crowding out of the glandular elements to add atrophy to the picture, sometimes with secondary nuclear changes in the surface epithelium to add superficial gastritis to the diagnosis, and sometimes with reactive hyperplasia of certain epithelial elements. Upon superficial consideration, there are convenient advantages to such a unitarian theory; it side-steps confusion over mixed and intermediate pictures, it simplifies etiologic problems, and it supplies a degree of continuity to one's thinking about gastritis in general, which is comforting, if nothing else. Whether on or several pathologic processes are at work, at the current stage of understanding it does not appear wise to accept the histopathologic diagnosis of gastritis if a relative increase in the degree of interstitial infiltration is the only unusual feature that can be demonstrated.

He who is not able to explain the gastroscopic picture of chronic hypertrophic gastritis by histopathologic abnormality must accept the responsibility for explaining the mechanism of the "diseased" mucosa's pebbled appearance, a striking feature of the endoscopic picture. Aparrently use of the term "etat mamelonne", which occurs frequently through the gastroscopic literature, has strayed far from its intended meaning because of failure of pathologic confirmation. In fact British gastroscopists have considered it to indicate a normal condition in which the areae gastricae are merely prominent because of increased depth of the delimiting creases. Results of the present observations permit no conclusion other than that the gastroscopic finding of pebbling indicates some unusual physiologic state of the areae gastricae. Possibly it is an abnormal tonic quality of the muscularis mucosae which is responsible, and perhaps somatic therapy is often effective because there are associated major motility or tonic abnormalities of the upper gastrointestinal tract in "chronic hypertrophic gastritis."

The significance of superficial gastritis appears to be associated largely with its two recognized sequelae, hemorrhage and atrophic gastritis.